Program/Abstract # 172
Examining the role of the *C. elegans* uterine Anchor Cell in vulva morphogenesis
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The *Caenorhabditis elegans* vulva is an excellent model system to explore the process of organ morphogenesis. The uterine Anchor Cell (AC) is vital to the coordination of development of the uterus and vulva. The AC has been well-studied for its involvement in complex signaling events that initiate cell specification in both the uterus and the vulva. The AC is also required to form a functional uterine–vulva connection by invading the basement membranes situated between the two organs and sitting between dorsal vulva cells, anchoring the uterus to the vulva. Recently, we have observed that the AC plays a role in the process of vulva morphogenesis, outside of its role in cell fate specification. Using several mutants, we have shown that when the AC fails to invade the vulva, fate specification of the vulva is largely normal, but the dorsal vulva structure and lumen formation is perturbed. Thus, AC invasion of the dorsal vulva is important to establish not only the uterine–vulva connection but also morphogenesis of the vulva. One caveat of our genetic approach is that these mutants also have uterine defects, so we are creating a system in which only the process of AC invasion is perturbed. This will allow us to understand the importance of AC invasion on vulva morphogenesis. Additionally, we are examining polarity of the vulva cells and their improper cell shape to understand why physical contact with the AC is needed to create the luminal structure of the dorsal vulva, but not the ventral vulva. Through the use of genetic, molecular and microscopic techniques, we hope to better understand the vital role of this cell invasion event in vulva morphogenesis.

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Program/Abstract # 173
Notch signaling and morphogenesis of single-cell tubes in the *C. elegans* digestive tract
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During organogenesis of the *Caenorhabditis elegans* digestive system, epithelial cells within a cyst-like primordium develop diverse and cell type-specific shapes through largely unknown mechanisms. We here analyze the morphogenesis of two adjacent epithelial cells in the cyst, called pm8 and vpi1, which become donut-shaped, or toroidal, single-cell tubes. pm8 and vpi1 delaminate from the dorsal epithelium and migrate across the cyst on a transient tract of laminin between ventral epithelial cells, while remodeling their apical junctions. pm8 appears to encircle the midline by wrapping around finger-like processes from neighboring cells. Finally, pm8 and vpi1 self-fuse to become toroids by expressing AFF-1 and EFF-1, respectively, two fusogens previously shown to promote cross-fusion between other cell types. Notch signaling is required for the expression of multiple genes, including *aff-1*, in pm8. In addition, Notch inhibits *eff-1* expression in pm8, thereby preventing cross-fusion between pm8 and vpi1. Thus, the tubulogenesis of pm8 and vpi1 involves highly orchestrated interactions with neighboring epithelial cells.

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Program/Abstract # 174
Mutagenesis screen in *C. elegans* suggests role of mor genes in pharyngeal development
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Achieving a clear understanding of the cellular and molecular phenomena driving organ formation is one of our lab's goals. In the present study we conducted a mutagenesis screen using a *Caenorhabditis elegans* pharyngeal muscle protein, myosin-2, tagged with green fluorescent protein as a visual assay. The screen produced over 200 pharyngeal mutant lines. Interestingly, 20 mutants manifested short and wide blunt pharynges, suggesting that genes required for embryonic elongation were mutated. Some blunt mutants were viable, while others died at the L1 stage, which may reflect the degree of defective elongation. To locate the alleles responsible for disrupting the elongation process we performed single nucleotide polymorphism (SNP) mapping. Thus far, we successfully linked 10 different mutant phenotypes to chromosomal regions. The gene, mor-1, which results in a shortened, rounded pharynx, was mapped to chromosome III. Furthermore, we found another 14 similar phenotypes, which may represent at least two other genes, mor-2 and mor-3. mor-2 has not been cloned, but is located on chromosome IV and has been shown to yield very similar phenotypes as mor-1. The mor-3 gene, a calcium/calmodulin dependent protein kinase may also be ascribed a role in abnormal pharynx development. Finally, sma-1, which is required for proper pharyngeal elongation, shares phenotypic similarity with some of our blunt mutants. We believe that these mor genes share the same molecular pathway and the remaining blunt phenotypes may be a result of defective morphogenesis. Understanding the pathway in which the mor genes work will yield a greater comprehension of pharyngeal morphogenesis.

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Program/Abstract # 175
Overcoming genetic redundancy to identify proteins acting in *C. elegans* gastrulation
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Morphogenesis is a critical but incompletely understood part of animal development. A major hurdle in the study of morphogenesis is the redundancy of genetic mechanisms involved. Morphogenesis genes have not fallen out of standard mutagenesis screens in diverse organisms as often as one might anticipate, and others have proposed that this is in part a result of genetic redundancy. *Caenorhabditis elegans* is an excellent model system for resolving problems of redundancy, since several techniques allow one to simultaneously disrupt the function of multiple genes in *C. elegans*. We have tested the hypothesis of redundancy in gastrulation, the first process of morphogenesis in the *C. elegans* embryo, by combining mutants with subtle effects on gastrulation and analyzing double mutants for synergistic effects. Our results to date suggest that redundant genes may contribute to the initiation of gastrulation, ingression of the E-